**Mycology**

**Opportunistic**

1. Microsporidiosis
2. Zygomycosis
   1. Rhinocerebral zygomycosis
   2. Mucormycosis
3. Candidiasis
   1. Chronic mucocutaneous
4. Pneumocystis PCP
5. Cryptococcus
6. Aspergillosis
   1. Necrotizing pseudomembranous bronchial aspergillosis

**Systemic Endemic (all dimorphic)**

1. Blastomycosis
2. Histoplasmosis
   1. African Histoplasmosis
3. Coccidioidomycosis
4. Paracoccidioidomycosis
5. Penicilliosis

**Subcutaneous (molds acquired through traumatic implantation)**

1. Sporothrichiosis
2. Chromoblastomycosis
3. Eumocotic mycetoma
4. SubQ Zygomycosis
5. SubQ Phaeohyphomycosis.

**Cutaneous (Hair, nails, skin)**

1. Dermatophytosis
   1. Tinea barbae
   2. T.capitis (favus)
   3. T.corporis
   4. T.cruris
   5. T.pedis (moccasin, interdigit, vesicular)
   6. T.unguium (prox, dist, superficial, candida)
2. Pityriasis versicolor

**Fungi**

Fungi are Eukaryotic organisms with a plasma membrane (additives like **ergosterol**) and an external cell wall (**chitin, glucan, mannin**). Symbiotic relationships: plants, insects, algae/cyanobacteria (lichens). Some are carnivorous and mecelial network used to ensnare nematodes in the soil. Their effects are witnessed in medicine in the form of poisons, infection in immunocompromised, primary infections and allergies/hypersensitivity.

* Alpha Amanitin (12-24hrs): inhib RNA pol II with fulminant hepatitis
* Muscarine (30min): PSL, cholinergic poisoning, pinpoint pupils, diarrhea/vomit
* Ergot: bizarre behavior
* Psychoactive mushrooms
* Fungal toxins like alcohol, oxalic acid, penicillin, cyclosporine A, citric acid

7 phyla of fungi but only three are super important clinically: **Blastocladiomycota** (infect all eukaryotic groups), **Zygomycota** (sugar and pin molds in black bread mold important in diabetics), **Ascomycota** (single celled yeasts like candida and pichia pneumocystis histoplsama, aspergillus), **Basidiomycota** (Cryptococcus in HIV).

Molds have apical growth with mycelium branching (distrib. by spores). Yeasts have cellular growth with budding (distrib. by fluid films). The chitin in the cell wall is developed similarly to cellulose and is a NAG chain. Wall gives the mold its rigidity which is necessary for forward pressure (chitin is in high concentration in .

Conidia are asexually produced, non-motile spores produced naked on the fungus. **Blastoconidia** are pushed out of the condiophore and have a new cell wall; **thallic** conidiogenesis comes from the remodeling of existing hyphae. Zygomycetes use sporangiospores from a sporangium. Sexually produced spores undergo meiosis with sporulation apparatus known as the teleomorph (ascospores by ascomycetes and zygospores by zygomycetes).

**Antifungals**

Need to be cidal, broad, high specificity, low side effects, low resistance, oral AND iv and cheap

**Azoles**: Block Ergosterol. Fluconazole (yeast with some CNS activity), Itraconazole (broader), Voriconazole (best azole for broad spectrum and is sometimes safer/more effective than Amp B for Aspergillus). **Resistance by efflux primarily… over expression of erg11 makes more pumps that aren’t as easily recognized.**

**Echinocandins:** caspofugin (Cidal, IV, low toxic, minimal CSF/urine, interact cyclosporin), anidulafugin (IV only with longest t1/2, no interactions), micafugin (fungicidal and potent for aspergillus), aminocandin… Work well for azole-resistant candida and act by targeting cell wall synth. **Resistance by FKS1 gene is mutated so it is no longer recognizable. Also the Cdr2p efflux pump may be involved. Resistance to one echinocandin does not infer resistance to all forms.**

**Polyenes:** Amphotericin B (conventional or lipid complex/liposomal/colloidal dispersion) and Nystatin. Biggest problem is nephrotoxicity but it hasthe broadest spectrum BY FAR. 40 years of use with little resistance. The drug punches holes in the membrane of the fungi. **Resistance by decreasing the total amount of ergosterol or by replacement of ergesterol by sterols with less affinity for polyenes or by masking the ergosterol so that thermodynamically/sterically unfavorable.**

**5 Fluorocytosine:** used to be an anticancer drug that can inhibit fungi (except filamentous fungi). **Reduced uptake of the drug due to loss of cytosine permease activity, or blocking the FUMP formation due to decreased cytosine deaminase or UPRtase activities.**

**Allylamines:** terbinafine (Lamisil) prevents the epoxide formation on squalene leading to increases in squalene. GREAT for nail infections by T. rubrum. **Recent resistance has been reported but oddly it is not the result of a genetic change or invasion by a new species… instead it seems to be individual host factor mediated (family history, age and previous fungal tx).**